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Stimulant Use, Associated Psychopathology, and Eating Disordered Symptomatology

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STIMULANT USE, ASSOCIATED PSYCHOPATHOLOGY, AND EATING DISORDERED SYMPTOMATOLOGY

by

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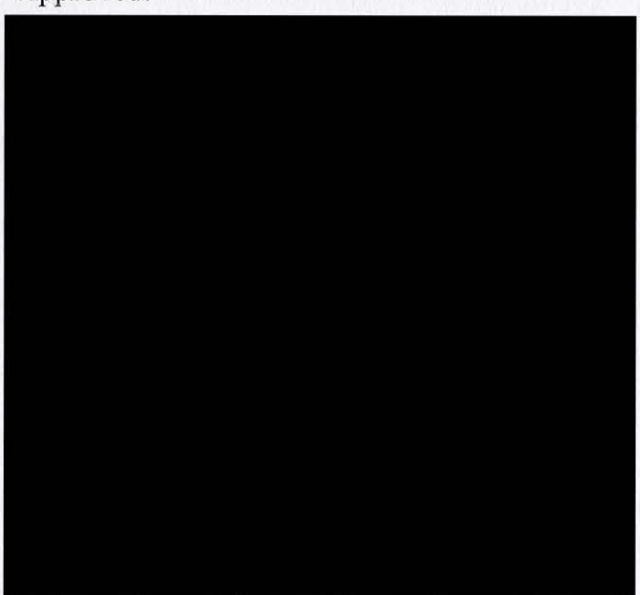

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ABSTRACT

STIMULANT USE, ASSOCIATED PSYCHOPATHOLOGY, AND EATING DISORDERED SYMPTOMATOLOGY

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The current study developed profiles of eating disorder, personality, and other psychopathological symptoms related to the use of central nervous system stimulants versus other types of drug use. Participants included 124 women in residential treatment for substance use with and without comorbid eating disorders. Symptomatology was measured by the Eating Disorders Inventory-3 (EDI-3), Millon Multiaxial Inventory-III (MCMI-III), and Personality Assessment Inventory (PAI). The current study utilized a series of six multivariate analyses of variance (MANOVAs) and discriminant analyses to determine patterns of psychopathology separating stimulant use from other drug use. Results indicated that women who used stimulants were primarily separated from women who used other drugs by scales measuring borderline personality pathology, and to a lesser extent, antisocial personality pathology. Stimulant users were also separated from other drug users by scales measuring diagnostic or associated features of borderline personality disorder (e.g., emotion dysregulation, suicide ideation, paranoia, aggression, drug use, and thought disturbance) and mood disturbance. Finally, stimulant users were differentiated from other drug users by a scale measuring extreme dieting and longing for thinness. Implications for treatment and future research are discussed.

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CHAPTER I

INTRODUCTION

Stimulant use is increasingly prevalent in recent decades. Women now equal men in stimulant use and demonstrate unique susceptibility to stimulant addiction due to hormonal interactions with stimulants (Greenfield, Back, Lawson, & Brady, 2010). The use of stimulants is of particular concern for those with eating disordered symptoms, as stimulant use is associated with decreased appetite, improved mood, and weight loss (National Center of Addiction and Substance Abuse (CASA) at Columbia University, 2003). Comorbidity between substance use disorders and eating disorders is associated with greater psychopathology and poorer prognosis than the individual diagnosis of either disorder (see review: Pearlstein, 2002). The current study assessed differences in personality, psychopathology, and eating disordered symptoms associated with the use of stimulants versus other types of drugs.

The background for the present study will be established by first reviewing the literature relating substance use disorders to eating disorders, then the literature linking substance use disorders to other forms of psychopathology, then the literature relating eating disorders to other forms of psychopathology. The integration of these three lines of research provide the basis for the present work. The current study addresses a void in the literature by examining clinical presentation based on type of substance use. Differences in clinical presentation will highlight the need to establish specific treatment modalities based on substance type, eating disorder symptomatology, and associated psychopathology.

Substance Use Disorders and Eating Disorders

Substance use disorders encompass both substance dependence and substance abuse. The Diagnostic and Statistical Manual (DSM-IV-TR; American Psychiatric Association (APA), 2000) defines the central elements of substance dependence as "a cluster of cognitive, behavioral, and physiological symptoms indicating that the individual continues use of the substance despite significant substance-related problems" (APA, 2000, p. 192). Individuals with substance dependence often manifest tolerance and withdrawal symptoms, desire to decrease substance use, and persist using the substance regardless of the negative physical and psychological effects caused by the substance. Substance abuse is described in the DSM-IV-TR as "a maladaptive pattern of substance use manifested by recurrent and significant adverse consequences related to the repeated use of substances" (APA, 2000, p. 198). Specifically, consequences may include difficulty fulfilling major role obligations, interpersonal issues, bodily harm, and legal ramifications.

Substance use disorders are categorized on the basis of substance type (APA, 2000). Central nervous system stimulants (hereafter: stimulants) include such drugs as nicotine, caffeine, methylphenidate (e.g. Ritalin), amphetamines (e.g., Adderall), methamphetamines (e.g. speed), and cocaine. Stimulants act on the mesocorticolimbic pathway of the brain, also known as the reward pathway, by increasing dopamine levels in the nucleus accumbens (Badiani, Belin, Epstein, Calu, & Shaham, 2011). Stimulant use is associated with a number of physiological responses, such as diminished appetite, hyper-alertness, improved concentration, and elevated mood; in greater amounts, it can result in such symptoms as rapid heart rate and breathing, sweating, tremor, and high

blood pressure (National Center of Addiction and Substance Abuse (CASA) at Columbia University, 2003; Weaver & Schnoll, 1999). Additionally, large, acute doses of amphetamine have been linked to aggression and transient psychotic symptoms, including paranoia, delusions, and hallucinations; the chronic use of amphetamines may result in persistent symptoms of psychosis and paranoia (reviews: Dawe, Davis, Lapworth, & McKetin, 2009; Shoptaw, Kao, & Ling, 2009).

The DSM-IV-TR (APA, 2000) outlines three eating disorder types and two eating disorder subtypes. Anorexia nervosa (AN) is diagnosed when "the individual refuses to maintain a minimally normal body weight, is intensely afraid of gaining weight, and exhibits a significant disturbance in the perception of the shape or size of his or her body" (APA, 2000, p. 583). Furthermore, the AN diagnosis can be subtyped into a restricting type (ANr) if the focus is on fasting and dieting, and a binge-eating and purging subtype (ANbp) when there is a cycle of binge-eating and purging (e.g., vomiting, laxative misuse). Criteria of bulimia nervosa (BN) include "binge eating and inappropriate compensatory methods to prevent weight gain" and "self-evaluation [which is] unduly influenced by body shape and weight" (APA, 2000, p. 589). Finally, EDNOS captures a wide variety of disordered eating, including binge-eating without purging (i.e., binge eating disorder) and subclinical AN or BN (APA, 2000, p. 594). Several studies examined associations between specific eating disorder types/behaviors and substance use.

Stimulant use and eating disorder symptomatology have numerous overlapping consequences, which may be more severe when the disorders occur comorbidly. Specifically, these two disorders consistently demonstrate some of the highest mortality

rates associated with psychological disorders (Arendt, Munk-Jørgensen, Sher, & Jensen, 2011; Crow et al., 2009), with the substance use-eating disorder comorbidity resulting in higher mortality rates than any other psychiatric comorbidity. Mortality is particularly related to substance misuse or overdose and suicide (Franko et al., 2005; Rosling, Sparen, Norring, & von Knorring, 2011). Additionally, both substance use and eating disorder symptomatology are associated with harmful and often irreversible health effects (Harrop & Marlatt, 2010; Greenfield, Gordon, Cohen & Trucco, 2010; Sansone & Sansone, 1994), pre- and posttreament impairments in quality of life (Hay & Mond, 2005, Tracy et al., 2012), increased social costs (e.g., treatment, job productivity) relative to normative populations (Meara & Frank, 2005), and increased health-related economic burden (Mitchell et al., 2009; Simon, Schmidt, & Pilling (2005). Additionally, there is an indication that those with particular combinations of comorbid diagnoses may have better outcomes associated with specific types of treatments. For example, Franko et al. (2005) found that recovery of alcohol use disorder with comorbid AN was best predicted by group therapy and hospitalization, whereas recovery from comorbid alcohol use disorder and BN was best predicted by individual therapy and exercise. Thus, an examination of these patterns of comorbidities may result in improved treatment outcomes and diminished consequences.

The literature is replete with studies examining the individual relationship between substance use disorders and eating disorders. Wiederman and Pryor (1996) described prevalence rates for substance use in the eating disorder population as ranging from 17% to 46% depending on the type and subtype of eating disorder. In a review of the literature, Holderness, Brooks-Gunn, and Warren (1994) found that of those

diagnosed with BN, up to 48.6% of individuals were also diagnosed with alcohol abuse or dependence, and up to 55% were diagnosed with any drug abuse or dependence. Similarly, across studies, they found that of individuals diagnosed with AN restricting type, up to 34% were diagnosed with alcohol abuse or dependence and up to 19% used illegal drugs. Finally, they reported that of individuals who were diagnosed with a combination of anorexic and bulimic symptoms, up to 45% also were diagnosed with alcohol abuse or dependence and up to 40% reported using street drugs. There is a clear and substantial relationship between substance use disorders and eating disorders.

Although individuals with eating disorders may use a wide range of substances, there appears to be a unique relationship between stimulant use and eating disordered symptomatology. The National Center of Addiction and Substance Abuse (CASA) at Columbia University (2003) found that smoking and eating disorder symptomatology were closely linked, in that nicotine suppresses appetite and provides an oral replacement to eating. They stated that women who smoke reference potential weight gain as a motivator for continued use; weight-linked motivation is cited by women at double the rate of men. Likewise, they found that women were more liable to resume smoking after quitting due to weight gain. Finally, in a study of college students, 39% of women and 25% of men initiated smoking as a tactic for dieting success (Office of the Surgeon General, 2001).

In a study of cocaine abusers, Cochrane, Malcolm, and Brewerton (1998) found that almost half of women presenting for cocaine abuse specifically used cocaine as a weight control measure; 72% of these were identified as meeting criteria for an eating disorder. Similarly, Jonas, Gold, Sweeney, and Pottash (1987) diagnosed eating disorders

in one-third of a sample of callers to the National Cocaine Hotline, via a structured clinical interview. In addition to appetite suppression, feelings of power and control initiated by cocaine have been identified as a motivator for use by women with eating disorders (National Center of Addiction and Substance Abuse (CASA) at Columbia University, 2003). Parks, Saewyc, Cox and MacKay (2008) found associations with stimulant use (i.e., amphetamines, cocaine, or cigarettes), disordered eating, and body dissatisfaction in the general population. In a sample of 30,000 British Columbian students, they found that individuals who had poor body image, binged, purged, or dieted, had a significantly higher likelihood of using stimulants than those who did not. Purgers were four times more likely to use stimulants than non-purgers; bingers, dieters, and those dissatisfied with their body were approximately twice as likely to use stimulants than their counterparts.

The stimulants methylphenidate and dextroamphetamine, both of which are used to treat Attention Deficit and Hyperactivity Disorder (ADHD), are also utilized for nonmedical purposes by individuals seeking weight loss. Williams, Goodale, Shay-Fiddler, Gloster, and Chang (2004) analyzed the misuse of these substances in 450 adolescents referred for treatment. They found that having an eating disorder and not attending school were the only two predictors which discriminated between stimulant use and other drug use; however, the effect size was small ($R^2 = .075$). Dukarm (2005) examined six individuals with bulimia nervosa and comborbid ADHD who were administered dextroamphetamine as a treatment for both. Patients self-reported a complete lack of bingeing and purging behaviors while on the drug. However, it was unclear as to whether the prescribed stimulant was truly alleviating the eating disorder

symptomatology, or if it was enabling more severe caloric restriction and thus extinguishing the binge-purge cycle.

Neurologically, Vicentic and Jones (2007) have tied cocaine- and amphetamine-regulated transcript peptides to both food and drug related rewards, which provide additional evidence to the link between stimulants and eating disorders. CART peptides regulate the mesolimbic dopamine system, which is associated with feeding and addiction. Although the temporal precedence of eating disorder symptomology and stimulant use has yet to be thoroughly investigated, the literature suggests that stimulant use may serve a particular purpose or function (e.g., appetite suppression) among individuals with body dissatisfaction or eating disordered symptomatology.

Specific eating disorder symptoms (e.g., restriction, bingeing, purging) demonstrate unique relationships to different types of substance use. Bulik et al. (1992) found that individuals with BN or ANbp had significantly higher use of cigarettes (Odds Ratio (OR) = 6.33), alcohol (OR = 6.61), laxatives (OR = 7.15), amphetamines (OR = 5.38), cocaine (OR = 7.03), and marijuana (OR = 4.54), relative to those with ANr. Purging, via laxative use, was associated with significantly higher use of emetics, marijuana, and amphetamines, when compared to non-laxative users. Wiederman and Pryor (1996) associated amphetamine use with increased caloric constraint; the study does not make causal claims; however, it may be that stimulant use enabled greater constraint. Purging was associated with cocaine, cigarette, and poly-drug use. Additionally, purgers were three and a half times more likely to use amphetamines and cocaine, and four times more likely to smoke, compared to non-purgers (Parkes, Saewyc, Cox, & MacKay, 2008). Finally, Piran and Robinson (2006, 2011) found that dieting and

purging were associated with the use of stimulants and abuse of sleeping medication, in two community samples. Given effect sizes, caloric constraint and purging, independently of bingeing, have a clear and strong relationship to stimulant use. These two eating disordered behaviors will be used to explore possible patterns of comorbidity associated with stimulant use.

Bingeing behaviors, unlike caloric constraint and purging, were generally associated with tranquilizer use, hallucinogens (Wiederman & Pryor, 1996), and alcohol use (Piran & Robinson, 2006; Wiederman & Pryor, 1996). Pure restriction was generally associated with less drug, alcohol, and psychotropic abuse or dependence than restriction with binge-eating and purging, or bingeing and purging alone (Corcos et al., 2001; Stock, Goldberg, Corbett, & Katzman, 2002). Therefore, purging and caloric constraint appear to be uniquely associated with stimulant use.

Substance Use and Associated Psychopathology

Historically, men were identified as having higher rates of substance use than women; however, recent clinical studies note a change in this trend. In a review of epidemiological surveys on substance abuse, Greenfield et al. (2010) reported that men continue to use cannabis, alcohol, heroin, and nicotine at higher rates than women. However, differences between genders have increasingly lessened over time. They noted similar rates of stimulant use between genders and evidence of equal or higher rates of nonmedical opioid use in women. The use of stimulants by women appears to be increasing more rapidly than other types of substances. This increase in stimulant use in women is evinced by tripled admission rates in federally funded treatment centers for pregnant women between 1994 and 2006, compared to doubled admissions for stimulant

treatment in the general population between 1995 and 2005. They also found associations between substance use and several psychological disorders (e.g. mood, anxiety, and eating disorders), with women who use substances having significantly more psychiatric diagnoses than men. These findings suggest that stimulant use is a growing problem among women and that patterns of comorbidity associated with stimulant use warrant further investigation.

Chen et al. (2011) examined psychiatric comorbidities associated with specific types of substance use by gender in an inpatient treatment sample (N = 465). In females, the most common comorbidity was cocaine dependence and "any other psychiatric disorder" (Male/Female odds-ratio = .54). Of women who used cocaine, comorbid diagnoses were mood disorders (32.6%), anxiety disorders (31.1%), psychotic symptoms (13.1%), Borderline Personality Disorder (27.7%), and Antisocial Personality Disorder (12.2%). These psychiatric comorbidity rates were greater than with any other type of drug.

Stimulant use was also associated with depression. Bohnert and Miech (2010) reported increases in the association between cocaine use and depressive disorders between the 1980's (Odds Ratio (OR) = 1.28) and 1990's (OR = 3.53). Of treatment-seeking cocaine users, depression was noted as one of the most frequent co-occurring psychological disorders (Kleinman et al., 1990; Rounsaville et al., 1991); further, many treatment-seeking users suffered from subclinical levels of depressive symptomatology. Additionally, depressed cocaine users reported greater euphoria associated with cocaine administration than nondepressed users, suggesting that cocaine use may be motivated or

maintained by a desire to alleviate negative emotions (Newton, Kalechstein, Tervo, & Ling, 2003; Uslaner, Kalechstein, Richter, Ling, & Newton, 1999).

Levental et al. (2010) reported that anhedonia, defined as diminished interest and pleasure in rewarding activities, had a particular relationship to stimulant use in a cross-sectional, population based sample (N = 43,093). Specifically, they found a distinct relationship between stimulant use or dependence with anhedonia and depressed mood across stimulant types (e.g. amphetamine, cocaine). Further, the relationship remained significant after controlling for demographic, psychiatric, and non-stimulant substance use characteristics. Odds ratio effect sizes were relatively large, as those using amphetamines were 3.31 times more likely to exhibit anhedonia, whereas those using cocaine were 2.56 times more likely to exhibit anhedonia. The effects were partially attenuated with the introduction of controls; however, they remained in the medium to high range.

Although these studies demonstrate an association between stimulant use and depression, there is a dearth of literature comparing depression severity among specific types of substance use disorders, with only one study directly comparing psychological comorbidities among different typologies of substances. Nevertheless, this literature suggests that stimulant users may display elevated rates of depression relative to other drug users.

Marken et al. (1992) found that marijuana and stimulants were the most commonly used illicit substances taken by inpatient individuals with manic symptoms. Similarly, Winokur et al. (1998) found that alcohol and stimulant abuse rates were significantly higher among bipolar groups than in unipolar or control groups. Stimulant

use is also being investigated as a potential treatment for bipolar disorder (Pagano, Demeter, Faber, Calabrese, & Findling, 2008; Wingo & Ghaemi, 2008). It is possible that those presenting with stimulant use may have elevations on scales measuring mania, though they may not actually present with bipolar disorder. Stimulant use and mania are associated with similar symptoms (e.g. decreased need for sleep, racing thoughts), and measures may not be able to differentiate between the two disorders.

Stimulant users also demonstrate elevations in anxiety sensitivity and anxiety disorders. Buckner, Proctor, Reynolds, Kopetz, and Lejuez (2011) found that anxiety sensitivity had a significant association to cocaine dependence; the relationship remained significant even when controlling for sex, age, alcohol dependence, hallucinogen dependence, major depressive disorder, panic disorder, and posttraumatic stress disorder. Herrero, Domingo-Salvany, Torrens, Brugal, & I.T.I.N.E.R.E. (2008) found that anxiety disorders were the second most common comorbidity (13%) among cocaine-users, surpassed only by mood disorders (26.6%; see also: Chen et al., 2011). Although there is a scarcity of research regarding the association between specific types of anxiety disorders and stimulant use, as well as the role of other types of substances, the literature indicates that stimulant users may have elevations in general measures of anxiety, relative to other types of drug users.

In a review of stimulant use and psychosis, Curran, Byrappa, and McBride (2004) found evidence for brief psychotic states brought on by large doses of stimulant drugs, which resolved within a few hours. They additionally found that the presence of positive symptoms of psychosis prior to stimulant use resulted in increased symptom severity upon initiation of stimulant use. However, the review noted two studies in which the

chronic use of injectable stimulants resulted in increased rates of chronic psychosis, with continued symptomatology long after stimulant use was discontinued (see also: Dawe et al., 2009; Shoptaw et al., 2009). Subsequent studies reported that 'chronic' or persistent psychosis among stimulant users was predicted by the early onset of stimulant use and extended duration of use (Chen et al. 2003; Lichlyter, Purdon, & Tibbo, 2011).

Certain personality traits may be conceptualized as risk factors for substance use disorders. Grekin, Sher, and Wood (2006) found that specific personality patterns were predictive of certain types of substance misuse. Namely, antisociality, novelty seeking, conduct disorder symptoms, and neuroticism were predictive of a variety of substance misuse. They additionally found unique personality patterns associated with alcohol, nicotine, and general drug use. For example, alcohol use symptoms were predicted by high extraversion and low openness, drug symptoms were predicted by low conscientiousness, and tobacco symptoms were predicted by high openness and low conscientiousness. Similarly, Kotov, Gamez, Schmidt, and Watson (2010) found that of the 'Big Five' personality traits, only disinhibition was associated with substance use. A high level of novelty seeking was associated with significantly greater stimulant use, with stimulant users being motivated by obtaining positive rewards (Adams et al., 2003). In a review of neuroimaging studies of stimulant users, Li and Sinha (2008) noted that stimulant use was associated with brain activity suggestive of impairments in cognitive inhibition and emotion regulation, as well as in increased impulsivity. In a review comparing stimulant and opioid addiction, Badiani et al. (2011) reported that in multiple animal studies, rats who later developed stimulant addictions demonstrated unique elevations in trait impulsivity prior to initiation of stimulant use; stimulant use

administration resulted in increased expression of impulsivity. The relationship with impulsivity was not found in opioid addicted rats.

Valila (2008) attempted to differentiate personality traits between participants who identified CNS stimulants, depressants, or opioids as their drug of choice. He found a large effect (Partial Eta Squared = .85) for drug of choice on personality trait scores. Specifically, participants who engaged in CNS stimulant use were significantly higher on the Extraversion domain, whereas CNS depressant and opioid users were higher on the Neuroticism domain than stimulant users. Thus, distinct personality traits are associated with specific types of substance use. Stimulant use will likely be associated with personality disorders in which reward seeking, extraversion, disinhibition, impulsivity, and emotion dysregulation are emphasized. When these personality traits are considered in the context of other cognitive and behavioral changes associated with stimulants (e.g., transient psychosis, paranoia, & aggression), the overall clinical picture is suggestive of borderline personality disorder.

Stimulant use is associated with specific personality disorders, namely, antisocial personality disorder and borderline personality disorder. Paim-Kessler et al. (2012) found that crack users presented with significantly higher rates of antisocial personality disorder, relative to both powder cocaine and other psychoactive substance users. Furthermore, Echeburúa, DeMedina, and Aizpiri (2009) compared personality disorders among individuals presenting with pure alcohol dependence and comorbid cocaine abuse and alcohol dependence. Individuals presenting with comorbid cocaine abuse had significantly higher rates of antisocial (21%), narcissistic (14.5%), and borderline (11.3%) personality disorders, relative to individuals with pure alcohol use. However,

borderline personality disorder, followed by other cluster B disorders, were also the most common personality disorders in all individuals presenting for substance use treatment (Ray, Primack, Chelminski, Young, & Zimmerman, 2011). Furthermore, Feske, Tarter, Kirisci, and Pilkonis (2006) found that borderline personality disorder was a significant predictor for multiple categories of substance use, including: any substance use disorder, alcohol use, drug use, heroin, cocaine, or poly-substance use. Antisocial personality disorder emerged as a partial mediator of the relationship between borderline personality disorder and substance use disorders. Thus, stimulant use is associated with increased rates of antisocial and borderline personality disorders across several studies; only one study found an association to narcissistic personality disorder. However, the high rates of these personality disorders across substance use types may obscure particular relationships.

The literature suggests that individuals who use stimulants, relative to other types of substances, may present with increased prevalence or severity of mood disorders, psychotic disorders, and borderline and antisocial personality disorders. Additionally, individuals using stimulants may have elevations in core symptoms of anxiety; however, the relationship between specific anxiety disorders and substance use disorders remains unclear. Of note, the preponderance of the research examined the relationships between only two individual constructs at a time, which prevents the identification of common clusters of disorders and those disorders which may demonstrate greater impact or relevance to individuals who use specific types of substances.

Eating Disorders and Associated Psychopathology

Given the strong link between stimulant use and particular eating disorder behaviors such as caloric constraint and purging (Parkes et al., 2008; Piran & Robinson, 2006 & 2011; Wiederman & Pryor, 1996), it is important to examine psychopathology associated with these behaviors, as it may influence the clinical pattern found in stimulant users. In a review of the literature, Pearlstein (2002) found that major depression and dysthymia were highly prevalent across eating disorder types and subtypes. In a separate review, O'Brien and Vincent (2003) found that the highest rates of depression occurred in the ANbp subtype; given that this subtype is defined, in part, by caloric constraint and purging, depression and stimulant use may be linked through eating disordered psychopathology (see also review: Casper, 1998). Purging, alone or with bingeing, was consistently linked to increased levels of depression when compared to other types of eating disorder behaviors (Garner, Garner, & Rosen, 1993). These findings give added support to the hypothesis that stimulant use will result in higher levels of depression than other drug use, given the association between stimulant use, purging, and caloric constraint.

In a review of the literature, Pearlstein (2002) reported that eating disorders have not generally been linked to bipolar disorder, citing only three studies in the last twenty years. In a few instances, elevations in bipolar II were linked specifically to BN; similarly, rates of eating disorders were found to be minimally elevated in a bipolar sample. Therefore, although stimulant use alone has associations with mania, the literature suggests that eating disorder symptomatology may not have a role in these findings.

Anxiety disorders, specifically obsessive-compulsive disorders and social phobias, were commonly associated with eating disorders and substance use disorders. Lifetime prevalence rates range from 20-55% in AN and 13-75% in BN (Bulik et al., 1992; Pearlstein, 2002). Social phobias appeared to occur equally in AN and BN, whereas obsessive compulsive disorder occurred more in AN (Pearlstein, 2002). For individuals in treatment for substance use disorders with eating disorder symptoms, posttraumatic stress disorder (PTSD) symptoms were significantly higher in those who reported bingeing (Cohen et al., 2010). Spindler and Milos (2007) found that binge-eating and purging behaviors were linked to increased anxiety (Odds Ratio = 1.90 – 2.22) and substance use (OR = 1.65 for vomiting, 1.89 for bingeing, and 4.23 for laxative use). Similarly, they found that dieting and fixation on being underweight was related specifically to anxiety disorders (OR = 4.23); weight and appearance concerns were concomitant with affective (OR = 1.81) and anxiety (OR = 2.77) disorders. Given the association of stimulant use with caloric constraint and purging, findings indicate that stimulant use may be associated with increased symptoms of anxiety. However, specific types of anxiety disorders may not demonstrate a consistent relationship with stimulant use.

The personality trait of obsessionality may serve as a protective mechanism against substance use disorders in individuals with eating disorders (Thompson-Brenner et al., 2008). Vitousek and Manke (1994) found that individuals diagnosed with AN were often identified as restrained, compliant and obsessional. Those diagnosed with BN were less consistent in personality, though affective instability and impulsivity were particularly common. In a review, Pearlstein (2002) found the previous associations as

well as stress reactivity, affective dysregulation, impulsivity, novelty seeking, low self-esteem and interpersonal sensitivity in those with BN (see also reviews: Lilenfeld et al., 2000; Wonderlich & Mitchell, 2001); those with AN were further associated with perfectionism and negative self-evaluation.

Personality disorders were likewise linked to specific eating disordered behaviors, which in turn have particular relationships to stimulant use (e.g. caloric constraint, purging). Borderline personality disorder was consistently linked to bingeing and purging behaviors in AN binge-purge type and BN, at rates ranging from 33% to 71% (O'Brien & Vincent, 2003; Pearlstein, 2002; Rosenvinge, Martinussen, & Ostensen, 2000). Similarly, Spindler and Milos (2007) found that binge-eating and purging behaviors were linked to increased cluster B personality disorders (OR = 2.12 – 2.65).

Rosenvinge et al. (2000) performed a meta-analysis of studies regarding personality disorders and eating disorders from 1983 to 1998. They found that cluster C personality disorders (i.e. dependent, avoidant, obsessive-compulsive) occurred in equal rates across eating disorders, at rates of approximately 45%. Additionally, they found that cluster A disorders (i.e. paranoid, schizoid, and schizotypal) had a higher prevalence in BN, at 27%, compared to 12% of AN patients. In cluster B disorders (i.e. narcissistic, borderline, antisocial, and histrionic), BN (44%) was likewise higher than AN (15%). In a review of the literature, Pearlstein (2002) reported that AN was most commonly associated with avoidant personality disorder, whereas BN was most commonly associated with borderline personality disorder. Additionally, borderline personality disorder was specifically associated with the purging subtype of EDNOS (e.g. Purging Disorder).

A few authors utilized multivariate analysis to examine psychopathology associated with specific eating disordered types and behaviors. Craig (1997) examined the personality and clinical scales of the MCMI-III in a group of eating disordered patients (N = 70). Discriminate analysis determined that the Dysthymia, Major Depression, and Thought Disorder scales discriminated the AN and AN binge/purge type from the BN and EDNOS groups (SCC = .41). Given that the ANbp subtype is partially defined by purging and caloric constraint, the Dysthymia, Major Depression, and Thought Disorder scales of the MCMI-III may also demonstrate elevations among stimulant users. Ciccolo and Johnsson (2002) identified three clusters of associations with eating disordered behaviors. The cluster associated specifically with purging, in isolation or with bingeing, had elevated levels of aggression and somatization, as well as lower levels of interoceptive awareness. Similarly, Garner et al. (1993) associated purging with greater levels of depression, panic disorders, anxiety, and suicide attempts. Given the association between purging and stimulant use (Piran & Robinson, 2006; 2011), these forms of psychopathology may be similarly elevated among stimulant users.

The literature suggests the eating disordered symptoms associated with stimulant use may be related to increased depression, general anxiety, thought disturbance, and borderline personality disorder, compared to eating disorder symptoms associated with other types of substances. As the vast majority of studies examined the confluence of two disorders at a time, despite the multiple comorbidities associated with stimulant use, the relative importance of a given disorder to the overall clinical presentation remains unclear.

The Present Study

There is considerable literature demonstrating associations between substance use, eating disorders, personality disorders, and other clinical syndromes, but almost always relating two groups of disorders at a time (e.g., eating disorders and substance use). There is also evidence in the literature that comorbidities between any pair of disorder groups predicts poorer prognosis and that various combinations may respond differently to treatment modalities. In treatment settings, patients may present with more than two comorbid disorders. For example, it is not uncommon for substance use to be comorbid with eating disorders, personality symptoms, depression, and anxiety. Such complicated cases can be very difficult to treat and even more challenging in the development of a case formulation. Identification of common clusters of comorbidity is a logical first step toward clarifying the complex relationships among these multiply comorbid cases, which may in turn allow identification of specific intervention strategies to more effectively treat specific comorbid combinations.

The present study examines differences in personality, psychopathology, and eating disorder symptomatology between women who use stimulants compared with women who use other types of drugs. In the literature, stimulant use has been associated with specific types of psychopathology and eating disorder behaviors (Table 1), particularly mood disorders, anxiety, psychosis, borderline and antisocial personality disorders, caloric constraint, and purging. Similarly, the eating disorder behaviors associated with stimulant use are associated with elevations in depression, anxiety, thought disturbance, and borderline personality disorder. Therefore, stimulant use is

predicted to be associated with higher overall levels of psychopathology and personality symptoms; specifically, stimulant users will have significantly higher mood and anxiety scores, thought disturbance/psychosis scores, and borderline and antisocial personality scores. Additionally, stimulant use is predicted to result in higher eating disorder pathology, and thus elevated scores on the EDI-3 risk composites and the associated psychological scale (i.e., Emotion Dysregulation).

Table 1

Summary of Reported Findings

	Stimulant use	AN	BN	BED	ANr	ANbp	Restrict	Binge	Purge
Stimulants			|||			|	||||||	|	||||||
Hallucinogen								||	
Alcohol		||	||||		|	||			|
Marijuana			||			|			|
Sedatives			|						
Tranquilizer			|					|	
Poly-drug use			|						
Depression	|||||||	|||||||	||||||||||||	||||	||	||||||			|||
Mania/ Bipolar	||||||			|					
Dysthymia				|	|||	|||			
Anxiety	|||	||||||	||||	||			|	|	||
Social Phobia		|||||||||	|||||||||						
OCD		|||||||||| ||||||||	|||||						
Panic Disorder								|	|
PTSD		|||	||||||					|	
Somatization									|
Psychosis/ Thought Dist.	|||||||||||||||				|	|			
Paranoia	||								
Borderline	||||		||			|			|
Antisocial	|||								
Other Clust. B	|		|						
Cluster A			|						
Cluster C		|	|						
Impulsivity	|||||||||		||	|					
Disinhibition	|||||||								
Emotion Dys.	||||||||		|||||						
Aggression									|
Reward Seeking	|								
Extraversion	|								

Table 1 (continued).

	Stimulant use	AN	BN	BED	ANr	ANbp	Restrict	Binge	Purge
Novelty Seeking	|		||						
Suicide Attempts									|
Aggression	|								
Concern with body/ weight	||								
Eating Disorder	|||||								

Thought Dist.: Thought Disturbance
Other Clust. B: Other Cluster B
Emotion Dys.: Emotion Dysregulation
Note: Individual studies are counted from each review, to prevent under or over examination of relationships

The current study is particularly valuable, as there is a dearth of literature regarding patterns of multiple comorbidity associated with particular types of substance use. Relatively few studies have examined differences in presentation based on substance use type, with even fewer studies examining these presentations multivariately. If expected differences are found, the current study may indicate the need to establish specific treatment modalities based on comorbid eating disorder and substance use combinations.

CHAPTER II

METHODOLOGY

Subjects

Subjects were a convenience sample of women at a residential treatment center for women with substance use disorders, with and without comorbid eating disorders. Although the current sample contained a portion of women with AN, BN, and EDNOS diagnoses, all of these women had the central feature of binge eating or purging. There were no pure restrictors in this sample. The target population for the current study was women who engage in substance use. Although all women were being treated for substance use disorders with or without comorbid eating disorders, not all women will meet the clinical level for both diagnoses. All data gathered were part of standard assessments given upon admission to the treatment facility. Subjects were from various locations across the United States, though all obtained treatment in the southeastern United States.

The present study included a mix of approximately 70 archival participants and approximately 50 voluntary participants, for a total of 124 female participants. Archival data was obtained between December 2009 and October 2012, and was de-identified at the treatment facility before being released for research. It was not feasible to contact these patients individually; however, permission was granted by two Institutional Review Boards (Appendix A) before utilizing data from these patients. All participants entering the study from November 2012 to March 2013 gave informed consent for their data to be utilized in the study, with the understanding that their participation, or lack thereof, would in no way affect their treatment.

As effect sizes were consistently large within the literature, effects were expected to be large within the current study. Power analysis indicated that with a large effect size, 92 to 106 participants would be sufficient to capture group differences.

Procedure

All assessments were given by a mental health clinician, as part of standard intake procedures. Furthermore, a certified addictionologist interviewed each patient regarding their substance use. A mental health clinician completed a clinical interview to determine diagnoses for each participant, using DSM-IV-TR (APA, 2000) criteria. An onsite chart-review was completed for each participant, within the security parameters of the facility. The chart review was utilized to determine diagnoses, frequency, intensity, duration, and type of substance use, and to obtain scores from the assessment instruments. Between November 2012 and March 2013, all patients were informed that with their consent, their data would be used for both research and treatment planning. Upon verbal agreement by the patient to participate in the study, the clinician signed and dated the informed consent to maintain the patient's privacy. This precaution was utilized as an additional security measure, ensuring that there was no possible data trail leading back to the patient's identity. The consent forms and data were stored separately in locked filing cabinets, within the security parameters of the institution.

Instruments

Three instruments will be utilized in the present study: the Eating Disorder Inventory-3 (EDI-3; Garner, 2004), the Personality Assessment Inventory (PAI; Morey, 1991) and the Millon Clinical Multiaxial Inventory-III (MCMI-III; Millon, Millon, Davis, & Grossman, 2009). The EDI-3 will be utilized to determine eating disordered

behaviors and associated symptomology, whereas the PAI and MCMI-III will be utilized to determine elevations of particular personality disorders and psychopathology (e.g. depression and anxiety disorders).

Eating Disorder Inventory-3

The EDI-3 is a 91 item self-report instrument which measures psychological and behavioral traits common to eating disorders. The instrument is designed to be used as an aide to diagnosis, in conjunction with clinical interviewing, and as an outcome measure and research tool (Garner, 2004).

Garner (2004) details three Eating Disorder Risk scales: Drive for Thinness, which measures terror of weight gain and yearning for thinness; Bulimia, which measures rumination and behaviors relating to binge eating; and Body Dissatisfaction, which examines displeasure with shape and body mass. Garner (2004) also details eight psychological scales: Low Self-Esteem, which measures negative self-appraisal and feelings of insecurity; Personal Alienation, which assesses an impoverished self-understanding; Interpersonal Insecurity, which measures reservation and distress in social circumstances; Interoceptive Deficits, which evaluates misperceptions in correctly identifying and reacting to emotional cues; Emotional Dysregulation, which assesses impulsivity, volatility, anger, and substance misuse; Perfectionism, which assesses personal and goal achievement; Asceticism, which evaluates self-denial and control; and Maturity Fears, which measures the wish to return to childhood and maintain a prepubertal façade (Garner, 2004).

Garner (2004) reported that the EDI-3 was normed both nationally and internationally for Anorexia Restricting, Anorexia Binge/Purge, Bulimia, and Eating

Disorder not Otherwise Specified, as well as for adult and adolescent clinical populations. Additionally, Podar and Alik (2009) assessed 301 participants differing on sex, age, diagnosis, language and ethnicity, and examined the factorial structure of the EDI subscales. They found almost indistinguishable structures across clinical and non-clinical, as well as Western and non-Western participants, indicating that the EDI-3 is generalizable across cultures.

Garner (2004) reported that the internal consistency coefficients ranged from the .80s to the .90s for the three Eating Disorder Risk scales and eight psychological scales, across the three normative groups and four diagnostic categories. Additionally, they reported the median test-retest coefficients for the Eating Disorder Risk scale as .95 and the Psychological scales as .93. Finally, they reported that validity was established through the use of factor analysis and intercorrelational studies with external eating disorder measures (e.g., the EAT-26 and BULIT-R) and with external measures of personality and psychopathology (e.g., the Rosenburg Self-Esteem Scale and MCMI-II). The EDI-3, therefore, should be a reliable and valid measure of eating disorder symptoms and psychological features in this group of substance using women in residential treatment.

Millon Clinical Multiaxial Inventory-III

The MCMI-III is a 175 item self-report measure, which is designed to be completed in 20 to 30 minutes and can be administered in an individual or group setting. The MCMI-III contains 27 scales: 24 Clinical scales, which are delineated based on severity and according to Axis I and Axis II disorders, as well as three Modifying Indices, measuring disclosure, desirability, and debasement.

Millon et al. (2009) delineate 11 scales measuring personality patterns: Schizoid, which measures detached, apathetic, and asocial characteristics; Avoidant, which measures caution and anxiety regarding possible social rejection; Depressive, which assesses chronic glumness, pessimism, and an inability to experience pleasure; Dependent, which measures passiveness and a need for guidance; Histrionic, which measures need for attention, fear of isolation, and relational manipulation; Narcisstic, which assesses egotism, arrogance, and willingness to engage in exploitation; Antisocial, which measures deception, impulsivity, and engagement in illegal activities for personal benefit; Sadistic, which assesses gratification from the degradation and violation of others; Compulsive, which evaluates perfectionism and discipline, fear of reproach, and antipathy towards others; Negativistic, which measures passive aggressiveness; and Masochistic, which assesses fostering of self-exploitation and self-debasement.

Furthermore, Millon et al. (2009) delineate three scales which measure severe personality pathology, which were devised to encompass increased deterioration in personality, including social and psychotic deficits. The Schizotypal scale measures intentional isolation, selfishness, emotional blunting, and emotional guardedness. The Borderline scale assesses emotional lability, uncertain self-image, and paradoxical interpersonal relationships. The Paranoid scale assesses pervasive suspicion and mistrust of others, recalcitrance, and inflexibility.

Millon et al. (2009) outline seven scales which measure moderately severe clinical syndromes: Anxious, which measures many forms of anxiety (e.g., specific phobias, somatic complaints, hypervigilance); Somatoform, which measures the expression of psychological complaints through body complaints; Bipolar: Manic, which

assesses manic symptomatology (e.g., agitation, impulsivity, and restlessness); Dysthymia, which evaluates apathy, chronic fatigue, social withdrawal, and anhedonia; Post-Traumatic Stress, which captures reaction to trauma and anxious arousal; Alcohol Dependence, which measures issues with alcoholism, recovery failures, and social consequences; and Drug Dependence, which assesses a recurrent or recent history of drug abuse, as well as impulsivity, and social and personal consequences.

Finally, Millon et al. (2009) outline three scales which measure severe clinical syndromes. The Thought Disorder scale assesses disoriented, schizophrenic-like symptoms, such as hallucinations, delusions, disorganized thought and emotional blunting. The Major Depressive scale measures severely depressed, hopeless, and suicidal behaviors and cognitions. Finally, the Delusional Disorder scale examines antagonistic and paranoid delusions, along with disturbed thinking.

Millon et al. (2009) noted that the MCMI-III underwent a three-step validation process, including 1) theoretical-substantive, 2) internal structure, and 3) external-criterion. The validation process was sequential, with items having to meet the criteria for each step before they could be evaluated in the next step. The revision process utilized several hundred clinicians who previously utilized the MCMI-II, across 26 states and Canada, for a total sample of 998 subjects. These subjects included individuals from inpatient and outpatient treatment centers, correctional facility inmates, and college student counselees.

Millon et al. (2009) reported that across the Clinical Personality Patterns, Severe Personality Pathology, Clinical Syndromes, and Severe Clinical Syndromes scales, internal consistency alpha levels ranged from .66 (Compulsive scale) to .90 (Major

Depression), with scores exceeding .80 for 19 of the 24 scales. Additionally, test-retest reliability was established through re-administration of the MCMI-III to 87 individuals, over 5-14 days. Stability coefficients range from .84 to .96 for these 24 scales. Finally, the MCMI-III was correlated with a number of external measures, including the Beck Depression Inventory, Symptom Checklist-90-Revised, MMPI-2, and Michigan Alcoholism Screening Test.

Personality Assessment Inventory

The PAI (Morey, 2007) is a 344 item, self-report inventory which measures adult psychopathology and personality. Morey (2007) describes 11 clinical scales, five treatment scales, 2 interpersonal scales and 4 validity scales, which together comprise the 22 scales of the inventory. For the purposes of this study, only the clinical scales and one treatment scale will be utilized. Results are reported as t-scores, with scores above 70 falling in the clinical range.

Morey (2007) delineates 11 clinical scales: Somatic Complaints, which measures a preoccupation with physical complaints; Anxiety, which measures cognitive, physiological, and affective symptoms of anxiety; Anxiety-Related Disorders, which assesses anxiety related to phobias, traumatic stress, and obsessive compulsive disorder; Depression, which evaluates cognitions, emotions, and physiological symptoms associated with depression; Mania, which measures irritability, grandiosity, and activity level; Paranoia, which measures persecution, resentment, and hyper-vigilance; Schizophrenia, which evaluates psychotic experiences, social detachment, and thought disorder; Borderline, which assesses affective instability, identity problems, and self-harm; Antisocial, which measures criminal behavior, stimulus seeking, and egocentricity;

and Alcohol Problems and Drug Problems, which measure substance use and dependence, as well as problems and consequences related to use.

Morey (2007) describes five treatment scales established to measure the willingness to engage in treatment as well as complicating factors related to treatment. For the purposes of this study, only the Suicidal Ideation scale (i.e., measuring suicidal ideation, plans) was used.

Morey (1996) reported that the PAI was developed for use in both normative and clinical populations, in adults aged 18 years or above. The standardization sample was representative and modeled after the United States census. The internal consistency alphas were a median of .81, .82, and .86, for normative, college, and clinical populations. In substance using populations, median alpha levels for the full scales were .78 for a methadone-using sample (Alterman et al., 1995) and .86 for an alcoholic sample (Schinka, 1995). In an eating disordered sample, the mean reliability of the full scales was .82 (Tasca, Wood, Demidenko, & Bissada, 2002). For temporal stability, the median test re-test reliability was .86 in the standardization sample, with a four week interval between the test and retest (Morey, 1996).

Morey (1996) accumulated data regarding convergent and discriminant validity correlates in order to establish the construct validity of the PAI scales. He reported correlations of the individual scales with more than 50 concurrent indices of psychopathology. Additionally, validity scales were developed to ascertain efforts towards impression management and strategic or careless responding. Inconsistency and careless responding was addressed through 1,000 computer simulations of random responses; 99.4% of these 'simulations' were identified by the scales. Finally, these

validity scales were correlated with other such scales on similar measures, with correlations ranging from .4 to .6.

Statistical Plan

The data obtained in the present study were analyzed using a combination of descriptive and inferential methods. The independent variable was dichotomous and measured the presence or absence of stimulant use, with the understanding that an absence of stimulant use still implied the presence of other types of drug use. Substance use was ascertained on the basis of the addictionologist's report, from the chart review. Due to the nature of the research, it was not possible to randomly assign participants to groups, nor was there a practical way to ensure equal group sizes.

The hypotheses for the study were analyzed using multivariate analysis of variance (MANOVA) and descriptive discriminant function analysis (DDA). MANOVA was used to determine if there were mean differences between the two substance use groups (i.e., stimulant use versus 'other drug' use) on the scales of the EDI-3, MCMI-III, and PAI. Descriptive discriminant function analysis (DDA) was utilized to further assess the ability of each set of scales to discriminate between the two substance use groups. In essence, it was a multivariate follow-up to the omnibus test of significance found from the MANOVA. Univariate analyses were examined following the discriminant analyses; however, the focus of the analyses will be on the results of the MANOVA and DDA in order to minimize Type 1 error (Stevens, 2002).

A number of statistical tests were used to ensure that statistical assumptions were met and to report the results of MANOVA and DDA. First, multivariate normality was assessed by ascertaining univariate normality (e.g., removing outliers, checking

histograms, measures of central tendency, and standardizing skewness and kurtosis values). Secondly, multivariate analysis was tested using the Mahalonbis's Distance statistic (Meyers, Gamst, & Guarino, 2006). Homogeneity of covariance matrices was tested using Box's test (Field, 2009) for MANOVAs; this assumption was further assessed in DDA by comparing the log determinants to ensure they were in the same ballpark (Huberty, 2002, pp. 587-588). With regards to univariate assumptions, homogeneity of variance was assessed using Levene's test; violations to this test were followed up with F_{Max} tests (Field, 2009). If a scale grossly violated this assumption, it was not be interpreted at the univariate level. Given the nature of this data, Pillai's trace was used as the multivariate test statistic as it is the most robust to potential issues with heterogeneity of variance and covariance (Meyers et al., 2006). Bonferroni corrections were employed for alpha levels for both multivariate ($p < .017$) and univariate analyses ($p < .001$) in order to guard against Type 1 error (Meyers et al., 2006). Partial eta squared was utilized as an effect size for the MANOVAs, eta squared was used as an effect size for the univariate analyses, and canonical r^2 was used as an effect size for DDA (Field, 2009). Finally, with regards to DDA, .30 was used as an interpretive cut-off for structure correlations (Finch, 2009; Tabachnick & Fiddell, 2001), unless there were severe violations in the data, in which case .50 was utilized to minimize error (Finch, 2009).

This study employed three planned analyses based on a priori hypotheses. Using the EDI-3, the Drive for Thinness, Bulimia, Body Dissatisfaction, and Emotion Dysregulation scales were entered as dependent/outcome variables in a MANOVA and DDA, with the stimulant use dichotomy as the independent variable. Secondly, using the PAI, the stimulant use dichotomy was again the independent variable, and the

Depression, Mania, Anxiety, Schizophrenia, Borderline, and Antisocial scales were entered as dependent/outcome variables in a MANOVA and DDA. Finally, using the scales of the MCMI-III, the stimulant use dichotomy was the independent variable, and the Anxiety, Major Depression, Dysthymia, Bipolar: Manic, Thought Disturbance, Borderline, and Antisocial scales were entered as dependent/outcome variables in a MANOVA and DDA. Three heuristic analyses were employed to further extend the understanding of the relationships between the constructs. The remaining scales of each measure were entered as dependent variables into three MANOVAs and DDAs, separated by measure.

All three measures were utilized, despite overlap, as each measure contained unique information and scales which were not captured by other measures. Therefore, utilizing the scales of both measures in a single analysis may have resulted in a scale from one measure taking all of the variance at a multivariate level and masking the contribution of a similar scale on another measure. Furthermore, inclusion of both broadband measures permitted comparison of results on overlapping scales, thus speaking to validity and allowing for examination of the clinical utility of these instruments.

CHAPTER III

ANALYSES AND RESULTS

Preliminary Analyses

Data were assessed for normality using measures of central tendency, histograms, skewness and kurtosis, and z-scores. Among the MCMI data, two participants had outlying scores across scales; therefore, these two participants were excluded. After this removal, skewness and kurtosis were calculated and standardized. The data evinced skewness outside the cut-off of ± 3.28 (Field, 2009) in six of the 11 EDI-3 scales, three of the 13 PAI scales, and 12 of the 24 MCMI-III scales. Kurtosis was outside the cut-off of ± 3.28 (Field, 2009) in two of the 11 EDI-3 scales, three of the 13 PAI scales, and two of the 24 MCMI-III scales. Therefore, several of the scales demonstrated a non-normal distribution, which was expected given the clinical population. Data could not be transformed, however, as data evinced both positive and negative skew, platykurtic, and leptokurtic distributions depending on the scale; these distribution differences were likely due to differences in base rates of particular disorders measured by the scales. Mahalonobis' distance was calculated as a measure of multivariate normality (Meyers et al., 2006). All participants fell below the critical value of χ^2 (df 49) = 85.35, p = .001, providing evidence for multivariate normality. Therefore, although some caution should be employed in interpreting these results due to violations to univariate normality, there was evidence supporting the assumption of multivariate normality.

Participants were excluded from analyses due to missing data or invalid profiles; participant totals ranged from 110 to 114, depending on analysis. Levene's test for equality of variance was completed for each scale (Table 2). All scales with significant

results were followed up with Hartley's F_{Max} test (Field, 2009). The critical value for the F_{Max} test, given the smaller of our two group sizes (N = 31) and $\alpha = .05$, was 2.63. The variance ratios for the following scales exceeded the critical values for the F_{Max} test: Suicidal Ideation (2.84), Anxiety (6.10), Sadistic (2.79), Negativistic (3.02), Masochistic (2.98), and Posttraumatic Stress (4.07). Although each of these scales violate the assumption of equality of variances, the Anxiety and Posttraumatic Stress scales demonstrated a gross violation and should not be interpreted at the univariate level. The remaining scales (i.e., Suicidal Ideation, Sadistic, Negativistic, Masochistic) should be interpreted with caution.

Table 2

Results from Levene's Test for Equality of Variance; All Scales

Dependent Variable	F	DF 1	DF 2	Sig.
EDI-3				
Drive for Thinness	1.89	1	111	.172
Bulimia	3.17	1	111	.078
Body Dissatisfaction	0.16	1	111	.689
Emotion Dysregulation	5.71	1	111	.019
Low Self Esteem	2.51	1	112	.116
Personal Alienation	8.08	1	112	**.005**
Interpersonal Insecurity	0.05	1	112	.829
Interpersonal Alienation	5.52	1	112	**.021**
Interoceptive Deficits	3.09	1	112	.082
Perfectionism	0.10	1	112	.757
Asceticism	1.99	1	112	.161
Maturity Fears	1.24	1	112	.269
PAI				
Anxiety	0.01	1	111	.926
Depression	0.79	1	111	.376
Mania	0.50	1	111	.483
Borderline	0.60	1	111	.441
Antisocial	0.39	1	111	.536
Somatic Concerns	4.04	1	111	**.047**
Anxiety Related Disorders	0.04	1	111	.848
Paranoia	3.27	1	111	.073

Table 2 (continued).

Dependent Variable	F	DF 1	DF 2	Sig.
Schizophrenia	9.83	1	111	**.002**
Alcohol Concerns	0.65	1	111	.421
Drug Concerns	0.06	1	111	.810
Aggression	2.39	1	111	.125
Suicide Ideation	6.79	1	111	**.010**
MCMI-III				
Anxiety	14.99	1	109	**.000**
Bipolar: Manic	2.76	1	109	.099
Dysthymia	0.92	1	109	.341
Thought Disorder	20.43	1	109	**.000**
Major Depression	0.12	1	109	.730
Antisocial	2.90	1	109	.091
Borderline	9.70	1	109	**.002**
Schizoid	6.79	1	109	**.010**
Avoidant	8.81	1	109	**.004**
Depressive	0.81	1	109	.370
Dependent	12.36	1	109	**.001**
Histrionic	1.39	1	109	.241
Narcissistic	4.02	1	109	**.048**
Sadistic	19.88	1	109	**.000**
Compulsive	1.353	1	109	.247
Negativistic	38.00	1	109	**.000**
Masochistic	11.05	1	109	**.001**
Schizotypal	9.26	1	109	**.003**
Paranoid	5.97	1	109	.016
Somatoform	1.77	1	109	.186
Alcohol Dependence	5.65	1	109	.019
Drug Dependence	7.08	1	109	**.009**
Posttraumatic Stress	16.85	1	109	**.000**
Delusional Disorder	0.31	1	109	.582

*Bolding denotes significance

With regards to drug use, the sample was comprised of 29.8% stimulant users, 68.5% alcohol users, 47.6% opioid/opiate users, 28.2% GABA agonists users, 22.6% marijuana users, and 8.1% 'other' users. Of stimulant users, 2.7% used only stimulants, 32.4% used stimulants and one additional substance, and 64.9% used three or more classes of substances. Of other drug users, 55.2% used one substance in isolation, 31.0% used two types of substances, and 13.8% used three or more classes of drugs. With

regards to eating disorders, 35.5% of the total sample was diagnosed with an eating disorder, according to DSM-IV criteria. With regards to comorbidities, 59.50% of stimulant users and 25.3% of all other types of drug users were diagnosed with a comorbid eating disorder.

Results, EDI-3

Per the a priori hypotheses, stimulant use was dichotomized as a grouping variable and the Drive for Thinness, Bulimia, Body Dissatisfaction, and Emotion Dysregulation scales were entered as dependent variables into a MANOVA. Covariance homogeneity was supported by Box's test, $M = 15.57$, $F(10, 18054) = 1.48$, $p = .140$. The multivariate model explained a significant portion of the variance in stimulant use, *Pillai's Trace* = 0.18 $F(4, 108) = 5.83$, $p < .001$, *partial* $\eta^2 = .18$. Subsequently, univariate analyses were examined. Stimulant use was associated with significantly higher Emotion Dysregulation, $F(1, 111) = 14.51$, $p < .001$, compared to other drug use; Drive for Thinness, $F(1, 111) = 10.42$, $p = .002$, and Body Dissatisfaction, $F(1, 111) = 3.93$, $p = .05$, approached significance but did not meet the $p < .001$. The Bulimia scale did not differentiate between stimulant use and other drug use, $F(1, 111) = 2.98$, $p = .087$. Means and standard deviations are presented in Table 3.

Table 3

A Priori Univariate Analyses, EDI-3

Dependent Variables	Stimulant Use (N=33)	Other Drug Use (N = 81)	Eta Squared
Drive for Thinness**	40.55/14.32	31.56/12.93	.08
Bulimia	47.09/11.28	43.63/8.63	.03
Body Dissatisfaction*	42.45/12.93	37.31/12.22	.03
Emotion Dysregulation***	56.76/11.14	49.86/7.57	.11

Note: *p < .05, **p < .01, ***p < .001

Descriptive discriminant analysis (DDA) was used to further assess the ability of the four eating disorder scales to separate the two grouping variables of stimulant use and other drug use. Log determinants were relatively equal (Group 1: 16.85, Group 2: 17.60, Error: 17.21), providing further evidence for the homogeneity of covariance matrices. One function was extracted from the data, $\Lambda = .82$, χ^2 (df 4) = 21.32, $p < .001$. The canonical r^2 was equal to .17, indicating that the discriminant function accounted for 17.7% of the variance.

Standardized canonical discriminant function coefficients (Table 4), which indicate the strength of a scale's unique contribution to a function, demonstrated that the function was principally predicted by Drive for Thinness, followed by the Emotion Dysregulation. Body Dissatisfaction negatively predicted the function. The structure correlations (Table 4) indicated that Emotion Dysregulation demonstrated the highest correlation with the function, followed by Drive for Thinness; Body Dissatisfaction and Bulimia were also correlated to the function, albeit at lower levels. Group centroids, the standardized means of the two grouping variables, were .72 for stimulant users and -.30 for other drug users. Therefore, stimulant users were characterized by a desire for thinness in conjunction with dysregulated emotions.

Table 4

A Priori Discriminant Analysis Results, EDI-3

Dependent Variables	Structure Matrix (Correlations)	Standardized Discriminant Function Coefficients
Drive for Thinness	.66	1.04
Bulimia	.35	-0.20
Body Dissatisfaction	.41	-0.48
Emotion Dysregulation	.78	0.74

Per the heuristic hypotheses, stimulant use was dichotomized as a grouping variable, and the remaining scales of the EDI-3 (e.g., Low Self-Esteem, Interoceptive Alienation) were entered as dependent variables into a MANOVA. Stimulant use did not account for a significant portion of the variance in the multivariate model, *Pillai's Trace* = 0.10, F (8, 105) = 1.40, p = .207. Due to this lack of significance, univariate analyses and discriminant analysis will not be further discussed for this model.

Results, PAI

In accordance with a priori hypotheses, the stimulant use dichotomy was entered as the independent variable and the Anxiety, Depression, Mania, Schizophrenia, Borderline, and Antisocial scales of the PAI were entered as dependent variables into a MANOVA. Covariance homogeneity was supported by Box's test, M = 25.338, F(21, 12470) = 1.11, p = .327. The multivariate model explained a significant portion of the variance in stimulant use, *Pillai's Trace* = 0.25, F (6, 106) = 5.75, $p < .001$, *partial* η^2 = .25. Univariate analyses indicated that stimulant use was associated with significantly higher scores on Borderline, F (1, 111) = 23.51, $p < .001$, and Antisocial scales, F (1, 111) = 21.65, $p < .001$, compared to other drug use. Depression, F (1, 111) = 6.03, p = .016, Mania, F (1, 111) = 7.54, p = .007, and Schizophrenia, F (1, 111) = 6.85, p = .010 approached significance but did not meet the $p < .001$ level. The Anxiety scale did not differentiate between stimulant use and other drug use, F (1, 111) = 2.15, p = .146. Means, standard deviations, and effect sizes are presented in Table 5.

Table 5

A Priori Univariate Analyses, PAI

Dependent Variables	Stimulant Use (N=31)	Other Drug Use (N = 82)	Eta Squared
Anxiety	66.84/14.10	62.51/13.96	.02
Depression*	71.81/15.28	64.61/13.35	.05
Mania**	54.35/10.71	48.73/9.32	.06
Schizophrenia**	59.65/16.35	52.68/10.92	.06
Borderline***	75.06/13.59	62.21/12.14	.18
Antisocial***	67.55/13.07	54.84/12.91	.16

Note: *p < .05, **p < .01, ***p < .001

DDA was used to determine the ability of the six personality and psychopathology scales to separate stimulant use from other drug use. Log determinants were relatively equal (Group 1: 26.09, Group 2: 26.63, Error: 26.47), providing further evidence for the homogeneity of covariance matrices. One function was extracted from the data, $\Lambda = .76$, χ^2 (df 6) = 30.42, $p < .001$. The canonical r^2 was equal to .24, indicating that the discriminant function accounted for 24.5% of the variance. Standardized canonical discriminant function coefficients (Table 6) indicated that the function was principally positively predicted by the Borderline scale, and to a much lesser extent, the Antisocial and Depression scales. Anxiety strongly negatively predicted the function; to a lesser extent, Schizophrenia also negatively predicted the function. The structure correlations (Table 6) demonstrated that Borderline and Antisocial evinced the highest correlations with the function, followed by Mania, Schizophrenia, and Depression. Group centroids were .92 for stimulant users and -.35 for other drug users. Therefore, the function characterizes stimulant users as elevated on personality disorders whose primary traits include impulsivity, irritability, and affective instability, and to a lesser extent, mood disturbance distinguished stimulant users from other drug users. The

role of the Schizophrenia scale is somewhat unclear, as it evinces a moderate correlation with the function but demonstrates a small but negative impact.

Table 6

A Priori Discriminant Analysis Results, PAI

Dependent Variables	Structure Matrix (Correlations)	Standardized Discriminant Function Coefficients
Anxiety	.24	-0.72
Depression	.41	0.30
Mania	.46	0.20
Schizophrenia	.44	-0.24
Borderline	.81	1.04
Antisocial	.77	0.30

With regards to the heuristic hypotheses, the stimulant use dichotomy was again entered as the independent variable, with the remaining seven scales of the PAI entered as dependent variables into a MANOVA. Covariance homogeneity was supported by Box's test, $M = 36.11$, $F(28, 11929) = 1.17$, $p = .245$. Stimulant use explained a significant portion of the variance in the multivariate model, *Pillai's Trace* = .16, $F(7, 105) = 2.90$, $p = .008$, *partial* $\eta^2 = .16$. Univariate analyses indicated that stimulant use was associated with significantly higher scores on Paranoid, $F(1, 111) = 10.76$, $p = .001$, and Suicide Ideation scales, $F(1, 111) = 11.52$, $p < .001$, compared to other drug use. Drug Problems, $F(1, 111) = 5.08$, $p = .026$, and Aggression, $F(1, 111) = 10.10$, $p = .002$, approached significance but did not meet the $p < .001$ level. The Anxiety Related Disorders scale, $F(1, 111) = 2.27$, $p = .135$, and the Somatization scale, $F(1, 111) = 1.62$, $p = .205$, did not differentiate between stimulant use and other drug use. Means, standard deviations, and effect sizes are presented in Table 7.

Table 7

Heuristic Univariate Analyses, PAI

Dependent Variables	Stimulant Use (N=31) Mean/SD	Other Drug Use (N = 82) Mean/SD	Eta Squared
Somatic Concerns	59.97/13.82	56.71/11.45	.01
Anxiety Related Disorders	65.19/13.73	60.98/13.11	.02
Paranoia***	60.68/14.71	52.32/10.96	.09
Alcohol Concerns	81.10/21.50	78.50/22.82	.00
Drug Concerns*	83.61/23.41	72.56/23.19	.04
Aggression**	55.32/13.31	47.41/11.27	.08
Suicide Ideation***	62.26/17.81	52.51/11.69	.09

Note: *p < .05, **p < .01, ***p < .001

DDA was used to further separate the stimulant group from the other drug use, using the six remaining PAI scales. Log determinants were approximately equal (Group 1: 35.92, Group 2: 36.96, Error: 36.53), providing additional evidence for the homogeneity of covariance matrices. One function was extracted from the data, $\Lambda = .84$, χ^2 (df 7) = 19.01, $p = .008$. The canonical r^2 was equal to .16, indicating that the discriminant function accounted for 16.2% of the variance. Standardized canonical discriminant function coefficients (Table 8) indicated that the function was primarily and equally predicted by the Suicide Ideation and Paranoid scales, and to a lesser extent, the Drug and Aggression scales. Anxiety Related Disorders slightly negatively impacted the function. The structure correlations (Table 8) demonstrated that Suicide Ideation, Paranoia, and Aggression had the highest correlations with the function, followed by Drug Use. Group centroids were .71 for stimulant users and -.27 for other drug users. Stimulant users are thus characterized primarily in terms of their suicidality, paranoia, and aggression, but may also be distinguished by drug use.

Table 8

Heuristic Discriminant Analysis Results, PAI

Dependent Variables	Structure Matrix (Correlations)	Standardized Discriminant Function Coefficients
Somatic Concerns	.28	-0.05
Anxiety Related Disorders	.33	-0.22
Paranoia	.71	0.49
Alcohol Concerns	.12	0.12
Drug Concerns	.49	0.27
Aggression	.69	0.28
Suicide Ideation	.73	0.55

Results, MCMI-III

Per the a priori hypotheses, the stimulant use dichotomy was entered as the independent variable, and the Anxiety, Bipolar: Manic, Dysthymia, Thought Disorder, Major Depression, Borderline, and Antisocial scales of the MCMI-III were entered as dependent variables into a MANOVA. Covariance homogeneity was not supported by Box's test, $M = 83.52$, $F(28, 14152) = 2.72$, $p < .001$; in conjunction with evidence of normality violations and unequal group sizes, these results should be interpreted with caution. The multivariate model explained a significant portion of the variance in stimulant use, *Pillai's Trace* = 0.943, $F(7, 103) = 3.31$, $p = .003$, *partial* $\eta^2 = .18$. Univariate analyses indicated that stimulant use was associated with significantly higher scores on Borderline, $F(1, 109) = 20.62$, $p < .001$, and Antisocial scales, $F(1, 109) = 14.26$, $p < .001$, compared to other drug use. Again, Bipolar: Manic, $F(1, 109) = 9.76$, $p = .002$, Dysthymia, $F(1, 109) = 4.26$, $p = .041$, and Thought Disorder, $F(1, 109) = 8.09$, $p = .005$, approached significance. The Anxiety scale was not interpreted due to violations in equality of variance. The Major Depression scale did not differentiate between

stimulant use and other drug use, $F(1, 109) = 2.21$, $p = .140$. Means, standard deviations, and effect sizes are presented in Table 9.

Table 9

A Priori Univariate Analyses, MCMI-III

Dependent Variables	Stimulant Use (N=33) Mean/SD	Other Drug Use (N = 78) Mean/SD	Eta Squared
Anxiety	83.12/12.40	71.15/29.31	.04
Bipolar: Manic**	67.09/22.76	50.76/26.12	.08
Dysthymia*	73.67/23.32	62.40/27.43	.03
Thought Disorder**	66.03/12.71	52.90/25.15	.07
Major Depression	64.88/33.06	54.60/33.38	.02
Antisocial***	79.91/15.65	63.95/22.01	.12
Borderline***	77.45/15.96	56.41/24.48	.16

Note: *p < .05, **p < .01, ***p < .001

DDA was used to determine the ability of the seven personality and psychopathology scales to separate stimulant use from other drug use. Log determinants showed some variability but were not grossly different (Group 1: 41.53, Group 2: 36.68, Error: 40.87), providing some evidence for the homogeneity of covariance matrices; however, caution should still be employed. One function was extracted from the data, $\Lambda = .82$, χ^2 (df 7) = 21.39, $p = .003$. The canonical r^2 was equal to .18, indicating that the discriminant function accounted for 18% of the variance. Standardized canonical discriminant function coefficients (Table 10) indicated that the function was almost entirely driven by the Borderline scale; the Major Depression scale negatively predicted the function. The structure correlations (Table 10) demonstrated that Borderline and Antisocial evinced the highest correlations with the function, followed by the Bipolar: Manic and Thought Disturbance scale. Given the violations found with this model, structure correlations were not interpreted below .5, in order to minimize error

(Dalgleish, 1994; Finch, 2009). Group centroids were .72 for stimulant users and -.31 for other drug users. With the exception of the contribution of the Major Depression scale, the results of the DDA using the MCMI-III scales were entirely consistent with the results of the DDA using the PAI scales above. Therefore, despite violations the function again distinguishes stimulant users as elevated on personality disorders whose criteria include impulsivity, irritability, and affective instability; to a lesser extent, mania, thought disorganization, and confusion distinguished stimulant users from other drug users.

Table 10

A Priori Discriminant Analysis Results, MCMI-III

Dependent Variables	Structure Matrix (Correlations)	Standardized Discriminant Function Coefficients
Anxiety	.46	-0.07
Bipolar: Manic	.63	0.08
Dysthymia	.42	0.02
Thought Disorder	.58	0.18
Major Depression	.30	-0.36
Antisocial	.76	0.29
Borderline	.92	0.82

With regards to the final heuristic hypothesis, the stimulant use dichotomy was entered as the independent variable, with the remaining 17 scales of the PAI entered as dependent variables into a MANOVA. It should be noted that this MANOVA is underpowered and corresponding discriminant analysis results may not be stable due to small sample size. Covariance homogeneity was supported by Box's test, $M = 231.57$, $F(153, 10000) = 1.57$, $p = .091$. Stimulant use explained a significant portion of the variance in the multivariate model, *Pillai's Trace* = .317, $F(17, 92) = 2.52$, $p = .003$, *partial* $\eta^2 = .32$. Univariate analyses indicated that stimulant use was associated with significantly higher scores on Sadistic, Negativistic, and Drug Dependence, compared to

other drug users. Conversely, other drug users score significantly higher on the Compulsivity scale, relative to stimulant users. Means, standard deviations, F-values, and effect sizes are presented in Table 11.

Table 11

Unplanned Univariate Analyses, MCMI-III

Dependent Variables	Stimulant Use (N=30) Mean/SD	Other Drug Use (N = 80) Mean/SD	F	Eta Squared
Schizoid	54.47/22.04	45.60/27.76	2.47	.02
Avoidant	59.60/26.67	52.73/31.78	1.11	.01
Depressive	67.33/26.45	62.23/27.11	0.79	.01
Dependent	78.70/19.03	69.04/26.59	3.32	.03
Histrionic	49.20/27.51	49.71/23.00	0.01	.00
Narcissistic	54.27/23.64	49.65/19.30	1.10	.01
Sadistic***	65.30/12.31	52.63/19.32	11.17	.09
Compulsive***	27.50/16.99	50.53/20.80	29.35	.21
Negativistic***	67.33/15.02	47.98/24.89	15.92	.13
Masochistic*	71.87/17.85	58.95/28.77	5.27	.05
Schizotypal*	55.37/22.54	44.25/27.26	3.97	.04
Paranoid*	53.40/24.57	39.75/27.35	5.73	.05
Somatoform	50.80/24.42	46.63/25.78	0.59	.01
Alcohol Dependence*	86.33/15.55	75.16/27.85	4.62	.04
Drug Dependence***	84.30/20.18	63.41/30.24	12.23	.10
Posttraumatic Stress**	66.20/11.66	52.49/25.96	7.75	.07
Delusional Disorder*	42.03/24.44	28.84/26.34	5.69	.05

Note: *p < .05, **p < .01, ***p < .001; df are 1 &108
Bolding denotes an inability to interpret due to violations to equality of variance

DDA was used to further separate the stimulant group from other drug use using the 17 remaining PAI scales. Log determinants were not equal (Group 1: 96.91, Group 2: 87.65, Error: 96.56), providing evidence against the homogeneity of covariance matrices. One function was extracted from the data, $\Lambda = .68$, χ^2 (df 17) = 37.99, $p = .002$. The canonical r^2 was equal to .32, indicating that the discriminant function accounted for 32% of the variance.

Due to violations in assumptions, .50 was used as an interpretive cut-off to minimize error (Dalgleish, 1994). Standardized canonical discriminant function coefficients (Table 12) indicated that the function was primarily predicted by the Histrionic, Negativistic, Avoidant, and Posttraumatic Stress scales. The Compulsive scale was a negative predictor of the function. The structure correlations (Table 12) demonstrated that the function was primarily positively correlated with Negativistic scale and negatively correlated with the Compulsive scale. There was some disagreement between the standardized coefficients and the structure correlations (e.g., Histrionic scale). Therefore, the structure correlations will be the focus of interpretation, as there is some evidence that these correlations evince increased stability in small sample sizes (Stevens, 2002). Group centroids were 1.10 for stimulant users and -.41 for other drug users. Therefore, the function characterizes stimulant users in terms of their passive-aggression and lack of compulsivity.

Table 12

Heuristic Discriminant Analysis Results, MCMI-III

Dependent Variables	Structure Matrix (Correlations)	Standardized Discriminant Function Coefficients
Schizoid	.22	0.35
Avoidant	.15	0.54
Depressive	.13	-0.76
Dependent	.26	-0.07
Histrionic	-.01	0.62
Narcissistic	.15	-0.00
Sadistic	.47	-0.21
Compulsive	-.77	-0.83
Negativistic	.56	0.55
Masochistic	.32	0.13
Schizotypal	.28	-0.11
Paranoid	.33	-0.15
Somatoform	.11	-0.33

Table 12 (continued).

Dependent Variables	Structure Matrix (Correlations)	Standardized Discriminant Function Coefficients
Alcohol Dependence	.30	-0.04
Drug Dependence	.49	-0.05
Posttraumatic Stress	.39	0.52
Delusional Disorder	.34	0.09

CHAPTER IV

SUMMARY

Discussion

Consistent with previous literature, women who used stimulants were separated from women who used all other forms of drugs on the basis of a relatively cohesive pattern of symptomatology. First and foremost, scales measuring traits linked with borderline personality disorder consistently and overwhelming separated stimulant users from other drug users. Additionally, scales measuring features of antisocial personality disorder consistently discriminated between stimulant users and other drug users, though these scales demonstrated less of an impact. These results are consistent with overlap between the diagnostic criteria of these two disorders, as they both feature difficulties with impulsivity, aggression, and emotion regulation (APA, 2000). Results are also consistent with findings of neuroimaging studies, which suggest that individuals addicted to stimulant drugs experience impairment in cognitive inhibition, emotion regulation, and impulsivity (review: Li & Sinha, 2008). The temporal precedence of stimulant use and these symptoms are still under investigation; however endophenotypic markers in humans (Ersche, Williams, Robbins & Bullmore, 2013) and trait studies in animals (Badiani et al., 2011) suggest that impairments precede stimulant use, but are exacerbated upon initiation of drug use.

Secondary to these personality types, stimulant users appeared to be separated from other drug users by mood disturbance. Both the PAI and MCMI-III separated stimulant users on the basis of manic symptoms. It was beyond the scope of this study to elucidate whether women experienced true symptoms of mania, or presented with manic-

like symptoms secondary to their stimulant use. Considering the rather paradoxical research investigating the use of prescription stimulants as a treatment for bipolar disorder (Pagano et al., 2008; Wingo & Ghaemi, 2008), it may be possible that women with mania were attempting to self-medicate their symptoms. It is equally possible that the measures utilized in the study had difficulty differentiating between the two presentations, given the overlap in symptoms. When using the MCMI-III, the average individual using stimulants reached the clinical range on the Bipolar: Manic scale; however, on the PAI, the average stimulant user was in the normative range on the Mania scale. Therefore, it may be that the PAI demonstrated better clinical utility in separating true symptoms of mania from those of stimulant use.

There was some evidence that depressive symptomatology also separated stimulant use from other drug use, although there was some conflict between measures. Results from planned analyses using the PAI indicated that depression was moderately correlated with stimulant use and had a small impact on predicting stimulant use, whereas results from the MCMI-III did not. Given the numerous violations of assumptions in the MCMI-III data, the results of the PAI should be accorded more weight. Additionally, the inclusion of both Dysthymia and Major Depression scales into the planned analysis using the MCMI-III may have diluted the results at the multivariate level and masked group differences. Therefore, consistent with previous research, women using stimulants may suffer from higher rates of depression and overall mood disturbance, but these clinical syndromes may play less of a role than severe personality pathology.

With regards to thought disturbance or psychosis, results suggested that stimulant users may experience slightly higher levels of symptomatology than other drug users.

However, scales measuring these constructs were correlated with functions measuring stimulant use, but either did not differentiate or made a small negative impact on functions separating stimulant use from other drug use. Thus, it may be that individuals experiencing thought disturbance are less likely to seek stimulants, particularly as the literature suggests that stimulant use may exacerbate positive symptoms of psychosis (reviews: reviews: Dawe et al., 2009; Shoptaw et al., 2009). Conversely, women who chronically use stimulants may find that they temporarily experience psychotic symptoms as a result of their use, but may not have insight into the relationship between stimulant use and psychosis. The average scores of stimulant users indicated that these women may experience "brief reactive psychosis" (Millon et al., 2009, p. 24) and may occasionally display inappropriate affect, disorganized thinking, and social isolation.

Exploratory analyses indicated that stimulant users were separated principally by elevations in scales measuring symptoms of suicidal ideation, paranoia, and aggression, and to a lesser extent, drug use. Additionally, stimulant use was defined by a passive-aggressive personality style, which was described as "vacillation between deference and defiance, between obedience and aggression," (Millon et al., p.18). Of these scales, only aggression was below the clinical range for experiencing significant symptom related distress or consequences. On the surface, these may appear to be a cluster of unrelated symptoms. However, each of these scales measure distinct diagnostic or associated features of borderline personality disorder (APA, 2000), giving additional weight to the premise that treatment-seeking women using stimulants may be primarily defined by borderline personality disorder. Specifically, these scales capture the following symptoms, as defined by APA (2000): "a pattern of … interpersonal relationships

characterized by alternating between extremes of idealization and devaluation," "impulsivity," "recurrent suicidal behavior, gestures, or threats," "affective instability," "inappropriate, intense anger or difficulty controlling anger," "transient, stress-related paranoid ideation," (p. 710) and the associated feature of "psychotic-like symptoms…during times of stress," (p. 708). Stimulant use may simultaneously aggravate symptoms of borderline personality disorder (e.g., impulsivity, aggression) and serve as a coping mechanism for mood dysregulation and affective disturbance.

These findings are largely consistent with the results of Chen et al. (2011), in that mood disorder, psychotic symptoms, borderline personality disorder, and antisocial personality disorder symptoms are highly problematic among women who use stimulant drugs. Furthermore, results are consistent with Feske et al. (2006), who reported that borderline personality disorder was a significant predictor for multiple categories of substance use, including poly-substance use. Within our sample, 97.3% of women using stimulants also used at least one other substance, compared to 44.8% of women using other types of substances.

Consistent with previous research, our sample of women presenting with stimulant use was saturated with comorbid eating disorder diagnoses (59.5%), compared to women presenting with other substance use disorders (25.3%). With regard to eating disordered symptomatology, women who used stimulants were separated from other drug users primarily by Emotion Dysregulation, a psychological scale posited to be associated with eating disorders, and Drive for Thinness. Although Emotion Dysregulation had a marginally higher correlation with the function defining stimulant use, Drive for Thinness overwhelmingly predicted stimulant use. Therefore, consistent with previous research,

stimulant use was associated with caloric constraint, extreme dieting, and a yearning for thinness, above and beyond other types of drug use. Additionally, it may be that the extreme dieting enacted by stimulant users interacted with their mood to exacerbate difficulties with regulating emotions. Previous research indicates that starvation is associated with symptoms mimicking mood disturbance (Casper, 1998; Pearlstein, 2002).

Given the physiological properties associated with stimulant use (e.g., decreased hunger, euphoria), it is possible that stimulant use served in a functional nature, in that it improved the individual's mood and ability to diet. Although this study was not causal in nature, the thin ideal espoused by women who used stimulants suggests that their stimulant use may have developed as a result of their motivation to lose weight, consistent with hypotheses in prior research (Baker, Mitchell, Neale, & Kendler, 2010; Holderness et al., 1994). Further research is needed to clarify the temporal precedence of stimulant use in women with eating disordered symptomatology.

Contrary to a priori hypotheses, stimulant users were not separated from other drug users by anxiety or anxiety related disorders. Stimulant users still appear to have difficulty with anxiety, as their average scores on anxiety and anxiety-related scales were suggestive of clinically significant symptoms. Given the link to borderline personality disorder, stimulant users may experience anxiety symptoms as one of numerous, intense mood states, thus explaining the elevations in scores. As women who used stimulants were also poly-substance users, they may seek different substances to alleviate particular mood states (e.g., stimulants for dysphoria, sedatives for anxiety). However, women presenting with pure anxiety disorders may seek other types of drugs, as the consistent heightened physiological arousal associated with anxiety may cause aversive reactions to

stimulants. Additionally, anxiety symptoms may partially result from stimulant use. For example, multiple animal studies have demonstrated elevations in anxiety in rats administered cocaine; studies suggested that consistent stimulant use resulted in anxiogenic effects (Erb, Kayyali, & Romero, 2006; Mantsch et al., 2008; Müller et al., 2008).

Finally, stimulant use was not separated from other drug use by purging behaviors, contrary to the a priori hypothesis. Given the extensive evidence in the literature for this relationship, the lack of findings in this study was likely related to limitations in instrumentation. This study did not have a pure measure of purging, but rather, a scale measuring the combination of bingeing and purging, as well as associated features such as drug use (i.e., the Bulimia scale). Therefore, many items on the scale may not have been relevant to the purging construct and may have masked important differences in purging behaviors between stimulant users and other drug users. The results of this study should not be interpreted as evidence against the stimulant use and purging relationship, particularly as constructs associated with purging were significant in the stimulant use profile.

Implications for Treatment

Given these findings, it is important to note that women seeking treatment for stimulant use disorders may present with a number of serious psychological problems. In such women, stimulant use may be secondary to longstanding personality pathology (i.e., borderline personality disorder) and eating disorder symptomatology. It will be necessary to carefully screen clients presenting for stimulant use and to triage their psychological and medical problems, focusing on those which may be life threatening (e.g., suicidality,

starvation associated with anorexia). If these features are present, the necessity for hospitalization should be carefully evaluated. Subsequently, a treatment plan should be developed, with the knowledge that an addiction focused treatment plan may not be effective in isolation, as it may not consider or treat the primary problems motivating stimulant use.

Given the findings of the current study, stimulant users may benefit from Dialectical Behavioral Therapy (DBT) in an effort to treat the symptomatology associated with borderline personality disorder (Linehan et al., 2006) and accompanying substance use (van den Bosch, Verheul, Schippers, & van den Brink, 2002); there is limited evidence regarding its effectiveness in treating eating disorders (e.g., Safer, Robinson, & Jo, 2010), although skills learned may present some benefit to women with eating disorders. DBT focuses on skill acquisition in the areas of distress tolerance, emotion regulation, interpersonal effectiveness, and mindfulness (Linehan, 1993).

Women presenting with comorbid stimulant use and antisocial personality disorder may benefit from motivational enhancement therapy (Miller, Zweben, DiClemente, & Rychtrik, 1992), to reduce their substance use and aid in treatment attendance and motivation. However, if stimulant users present with primarily eating disorder symptomatology, cognitive behavioral approaches (e.g., cognitive behavioral therapy for eating disorders; Fairburn, 2008) should be considered in order to target the underlying desire for thinness and weight control. It is important to note that the results of the current study are isolated to women and these treatment implications may not be generalizable to men who seek treatment for stimulant use.

Limitations of the Current Study

The current study had several limitations. Due to time constraints, it was not possible to collect more than 124 participants, so power may have been a limitation, particularly in the exploratory analyses which utilized a large number of scales. Additionally, several scales within the data demonstrated a non-normal distribution, which could not be corrected due to inconsistencies in the directionality of the skewness and kurtosis. Although multivariate tests for normality were within an acceptable range, the lack of equality between cell sizes, in conjunction with the lack of univariate normality in a number of the scales, indicate that these results should be interpreted with caution as the combination of these two violations can result in an overly liberal *F* statistic (Meyers et al., 2006).

Additionally, all of the measures utilized in this study were self-reported, which introduced the opportunity for bias via impression management and misunderstanding. Attempts were made to control for bias where possible (e.g., use of validity scales, presence of the patient's mental health clinician). However, the study would have benefited from collateral sources of reporting, clinician based measures, or behavioral monitoring, such as ecological momentary based assessment.

The current study may also have been hindered by uncontrolled, third variables. As the study examined only the presence or absence of stimulant use, women in both groups may have used a multitude of other drugs. This approach to the data increased confidence in the external validity of the results, as many individuals seeking treatment for alcohol and drug use present with poly-drug use, a premise which was supported by our data. However, this approach failed to account for the possibility that a different drug

may have been responsible for the group differences or masked true differences. Additionally, other, unmeasured constructs besides substance use may have accounted for the group differences in personality and psychopathology. The overlap in findings between the current study and the literature, however, do not provide evidence of a third variable problem.

Finally, the generalizability of the results are somewhat limited in this study, given the lack of randomization, use of an accessible population, lack of anorexia nervosa, restricting subtype diagnoses, and the complete lack of male participants. As the entire sample is from a treatment center in the southeastern United States, the sample may be unrepresentative of the general population or may be heterogeneous in some way. Furthermore, it was beyond the resources of this study to collect a sample of pure stimulant users. Although the current study offers much in the way of ecological validity, results of stimulant users who are also poly-drug users may not generalize to pure stimulant users.

Future Directions

Future studies should investigate potential gender differences in the clinical presentation of individuals with stimulant use disorders, as this study used only women participants. Furthermore, future studies should attempt to include a full range of eating disorder diagnoses (e.g., ANr), as well as both pure and poly-drug stimulant users, to more fully tease apart differences in clinical presentation associated with various patterns of comorbidity. Additionally, the results of this study should be replicated in a larger study, as certain exploratory analyses lacked the power to adequately rule out type I and II error. Furthermore, although numerous studies hypothesized the causal relationship

between stimulant use, emotion regulation, and eating disorder symptomatology, the literature would benefit from a well-controlled longitudinal study to examine the nature of these relationships.

APPENDIX A

INSTITUTIONAL REVIEW BOARD NOTICE OF COMMITTEE ACTION

THE UNIVERSITY OF SOUTHERN MISSISSIPPI

THE UNIVERSITY OF
SOUTHERN MISSISSIPPI.

INSTITUTIONAL REVIEW BOARD
118 College Drive #5147 | Hattiesburg, MS 39406-0001
Phone: 601.266.6820 | Fax: 601.266.4377 | www.usm.edu/irb

NOTICE OF COMMITTEE ACTION

The project has been reviewed by The University of Southern Mississippi Institutional Review Board in accordance with Federal Drug Administration regulations (21 CFR 26, 111), Department of Health and Human Services (45 CFR Part 46), and university guidelines to ensure adherence to the following criteria:

- The risks to subjects are minimized.
- The risks to subjects are reasonable in relation to the anticipated benefits.
- The selection of subjects is equitable.
- Informed consent is adequate and appropriately documented.
- Where appropriate, the research plan makes adequate provisions for monitoring the data collected to ensure the safety of the subjects.
- Where appropriate, there are adequate provisions to protect the privacy of subjects and to maintain the confidentiality of all data.
- Appropriate additional safeguards have been included to protect vulnerable subjects.
- Any unanticipated, serious, or continuing problems encountered regarding risks to subjects must be reported immediately, but not later than 10 days following the event. This should be reported to the IRB Office via the "Adverse Effect Report Form".
- If approved, the maximum period of approval is limited to twelve months. Projects that exceed this period must submit an application for renewal or continuation.

PROTOCOL NUMBER: **12032105**
PROJECT TITLE: **The Association of Personality and Psychopathology with Eating Disorder Type and Symotomology**
PROJECT TYPE: **Thesis**
RESEARCHER/S: **Tiffany Hopkins**
COLLEGE/DIVISION: **College of Education & Psychology**
DEPARTMENT: **Clinical Psychology**
FUNDING AGENCY: **N/A**
IRB COMMITTEE ACTION: **Expedited Review Approval**
PERIOD OF PROJECT APPROVAL: **04/17/2012 to 04/16/2013**

Lawrence A. Hosman, Ph.D.
Institutional Review Board Chair

FORREST GENERAL HOSPITAL

ForrestGeneral HOSPITAL

APPROVAL OF RESEARCH

October 18, 2012

Tiffany Hopkins, B.S.
Psychology Clinic
University of Southern Mississippi
118 College Drive #5025
Hattiesburg, MS 39406-0001

Dear Ms. Hopkins:

On October 18, 2012 the IRB chairman reviewed the following protocol:

Type of Review:	*Expedited Review*
Project Title:	*Stimulant use, associated psychopathology and eating disordered symptomatology*
Principle Investigator:	*Tiffany Hopkins, B.S.*
IRB ID:	*12-006*
Funding Agency:	*NIA*
Documents Reviewed:	*-Application for Human Research* *-Protocol #12022105* *-CITI Training for Tiffany Hopkins, B.S., Bradley Green, Ph.D. and Cathy Reed, Ph.D.* *-Informed Consent dated September 21, 2012* *-HIPPA IRB Waiver of Authorization*

This is to confirm that I have reviewed and approved the protocol and study related documents as submitted. You are granted permission to conduct your study as described in your application effective immediately. The study is subject to continuing review on or before 6/30/2013 unless closed before that date.

Attached is a stamped approved consent document. Use copies of this document to document consent.

Please note that any changes to the study as approved must be promptly reported and approved. Contact Michele Stanley at 601-288-4324, if you have any questions or require further information.

Sincerely,

Lewis E Hatten

Lewis E. Hatten, M.D.
Chairman, Institutional Review Committee

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